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NOAC BUT NOT VKA FAVORABLY AFFECT ENDOGENOUS THROMBOLYTIC STATUS: A NOVEL MECHANISM OF ACTION

Poster Contributions

Hall C

Saturday, March 29, 2014, 10:00 a.m.-10:45 a.m.

Session Title: Arrhythmias and Clinical EP: State of the Art Anticoagulation for Atrial Fibrillation

Abstract Category: 4. Arrhythmias and Clinical EP: AF/SVT

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Background: Patients with atrial fibrillation (AF) are at increased risk of stroke through thrombus formation. The propensity for thrombus formation is determined by the balance between prothrombotic factors and endogenous thrombolysis. Endogenous thrombolytic status is frequently impaired in patients with acute myocardial infarction, stroke, and renal failure. We sought to assess the effect of novel oral anticoagulants (NOAC) on global thrombotic and thrombolytic status, compared to vitamin K antagonists (VKA).

Methods: We tested global thrombotic status in patients with AF before and during treatment with NOAC (dabigatran n=12, rivaroxaban n=9) and compared them to patients with AF before and during treatment with VKA (n=13). The CHA2DS2VASc scores were similar in the NOAC and VKA groups (2.81.6 vs. 3.12, P=0.636). Thrombotic status was assessed by testing a native blood sample using the point-of-care Global Thrombosis Test (GTT). This automated test employs non-anticoagulated blood to assess thrombotic and thrombolytic status, by measuring the time required to form a shear-induced thrombus under physiological conditions (occlusion time, OT), and in the second phase of the test, measuring the time to achieve endogenous lysis of the thrombus (lysis time, LT).

Results: NOAC prolonged OT [median 483s (25th -75th %ile: 395-556) vs. 714s (553-842), P<0.0001] and reduced LT [1519s (1137-1927) vs. 724s (280-1592), P=0.032] compared to baseline. VKA prolonged OT [437s (313-550) vs. 639s (575-644), P=0.007] but had no effect on LT [1490s (1386-3368) vs. 1778s (1493-2365), P=0.724]. In the VKA group, INR at the time of testing was 2.50.6.

Baseline thrombotic status was similar in the NOAC and VKA groups [OT 483s vs. 437s, P=0.386; LT 1519s vs. 1490s, P=0.326]. Rivaroxaban and dabigatran prolonged OT similarly (P=0.651) and reduced LT similarly (P=0.917).

Conclusion: NOACs affect global thrombotic status differently from VKA. Although both prolong occlusion time, only NOACs favourably improve endogenous thrombolysis. This may contribute to the observed superiority of some NOACs in reducing thrombotic events and needs to be further evaluated in larger cohorts.